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EXAMINER

CHAPPELL, CHERIE M

ART UNIT

PAPER NUMBER

1647

DATE MAILED: 11/14/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

<b>Office Action Summary</b>	Application No. 10/694,579	Applicant(s) MEHTA ET AL.	
	Examiner Cherie M. Chappell	Art Unit 1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

### Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

### Status

- 1) ☒ Responsive to communication(s) filed on 8/12/2005.
- 2a) ☐ This action is **FINAL**.                      2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

### Disposition of Claims

- 4) ☒ Claim(s) 1-16 is/are pending in the application.
- 4a) Of the above claim(s) 8 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-7 and 9-16 is/are rejected.
- 7) ☒ Claim(s) 9 is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

### Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All    b) ☐ Some \*    c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.

### Attachment(s)

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)                        | 4) <input type="checkbox"/> Interview Summary (PTO-413)                     |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)               | Paper No(s)/Mail Date. _____  |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| Paper No(s)/Mail Date <u>4/07/2005</u>   | 6) <input type="checkbox"/> Other: _____                                    |

## DETAILED ACTION

### *Formal Matters*

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 12 August 2005 has been entered. Claims 1-16 are pending. Claim 8 has been withdrawn. Claims 1-7 and 9-16 are under examination. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.
2. This application contains claim 8 drawn to an invention nonelected with traverse filed 9/28/2004. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.
3. Applicant's IDS submitted on 04/07/2005 is acknowledged and has been considered. A signed copy is attached hereto. However, item C33 contained in the IDS has not been considered. The listing of references in the Search Report is not considered to be an information disclosure statement (IDS) complying with 37 CFR 1.98. 37 CFR 1.98(a)(2) requires a legible copy of: (1) each foreign patent; (2) each publication or that portion which caused it to be listed; (3) for each cited pending U.S. application, the application specification including claims, and any drawing of the application, or that portion of the application which caused it to be listed including any claims directed to that portion, unless the cited pending U.S. application is stored in the Image File Wrapper (IFW) system; and (4) all other information, or that portion which caused it to be listed. In addition, each IDS must include a list of all patents, publications, applications, or other information submitted for consideration by the Office (see 37 CFR 1.98(a)(1) and (b)), and MPEP § 609.04(a), subsection I. states, "the list ... must be submitted on a separate paper." Therefore, the references cited in the Search Report have not been considered. Applicant is advised that the date of submission of any item of information or any missing element(s) will be the date of submission for purposes of determining compliance with the requirements based on the time of filing the IDS, including all "statement" requirements of 37 CFR 1.97(e). See MPEP § 609.05(a).

*Claim Rejections Maintained*

*35 USC § 112 – First Paragraph, Scope of Enablement*

4. The rejection of claims 1 and 5 under 35 U.S.C. 112, first paragraph, for scope of enablement is maintained for the reasons of record.

5. Claims 2, 3, 4, 6, 7, 9, and 10 are also rejected under 35 U.S.C. 112, first paragraph, for the reasons of record and for the reasons which follow.

6. Claim 2 is also rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a change in myocardial wall thickness after an infarct and administration of G-CSF, calculated as average thickness from three short axis views in the area of maximum infarction, does not reasonably provide enablement for the use of G-CSF for a reduction in wall thickness losses. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with this claim. The claim recites the method of claim 1 wherein the reduction in damage is characterized by reduction in wall thickness losses. Example 1 in the specification teaches that G-CSF promotes myocardial repair by improving wall thickness in the infarct zone (p. 20, lines 7-8). However, the specification does not teach the use of G-CSF for a reduction in wall thickness losses. The loss of cardiac wall thickness (in cell numbers) happens as a direct result of the infarct. It is well known in the art that normal repair after an infarct includes cardiomyocyte hypertrophy – a process by which cardiac myocyte cell thickening occurs (see i.e. Melillo *et al.*, Circulation 1996 93:1447-58, specifically at third full paragraph of introduction [beginning at the bottom of page 2 of 24 as attached]; and definition of “hypertrophy” Stedman’s Medical Dictionary 27<sup>th</sup> Edition, 2000 Lippincott Williams & Wilkins). Cardiac hypertrophy naturally compensates for cell loss by increasing cell size. Thus, as a result of cardiomyocyte hypertrophy, cardiac wall thickening occurs. While the specification teaches that G-CSF promotes myocardial repair by improving wall thickness in the infarct zone, it does not provide support for “a reduction in wall thickness losses”. A reduction in wall thickness losses can only result from preventing or decreasing cardiac damage before or during an infarct. Applicant’s claims are directed to the improvement in a method of reperfusion therapy for treating an acute myocardial infarction (AMI). Thus, the reperfusion therapy method would not take place until after an AMI has occurred. If an AMI has occurred, wall thickness losses (in terms of cell numbers) have already occurred. The disclosure does not teach a method of

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preventing or decreasing cardiac damage before or during an infarct. Therefore, a claim to a reduction in wall thickness losses cannot be made within the scope of the disclosure.

7. Claim 10 is also rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for an improvement in a method of bypass surgery that reduces tissue damage consisting of administering an effective amount of a composition comprising Granulocyte Colony Stimulating Factor (G-CSF), does not reasonably provide enablement for a method of bypass surgery that would reduce tissue damage. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims. The disclosure fails to teach the steps of a method of bypass surgery that would reduce tissue damage. Bypass surgery may mitigate against further or future tissue damage, but it does not have an effect on already damaged tissue. The administration of a composition comprising G-CSF may aid in tissue repair after administration, but it does not limit damage that has already occurred. Therefore, a reduction of tissue damage cannot be made within the scope of the disclosure.

#### *Response to Amendments/Arguments*

8. Applicant's response filed 12 August 2005 fails to address the Examiner's final rejection under 35 U.S.C. 112, first paragraph - enablement. However, in an effort to promote compact prosecution, Applicant's arguments filed on 5/20/2005, with regard to prior rejections made under 35 U.S.C. 112, first paragraph, have been fully considered and are addressed *infra*.

9. Applicant's arguments, see Amendments and Arguments filed 08/12/2005, with respect to claims 1 and 5, have been fully considered, but are not persuasive. In response to Examiner's rejection that Applicant is not enabled for IL-8 and other art-known pro-inflammatory cytokines to reduce myocardial infarct-related damage, Applicant argues that the upregulation in IL-8 reported by Vallely *et al.*, could have been the result of an expansion of the total bone marrow cell number after surgery and a subsequent release of stem cells into the blood circulation. Applicants rely on Gzrelak *et al.*, (1998 Eur. Surg. Res. 30:198-204) to support this assertion (see Applicant's arguments of 5/20/2005, pp. 7-9) Applicant also states that Gzrelak *et al.*, and Laterveer *et al.*, (1996 Exp. Hematol. 24:1387-1393) teach away from IL-8's pro-inflammatory properties and instead teach that cytokines such as IL-8 may be useful in the

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mobilization of progenitor cells to mediate myocardial infarct-related damage. Applicant's arguments are not persuasive.

10. Applicant misapplies the teachings of Gzrelak *et al.* and Laterveer *et al.* Gzrelak *et al.*, teach a rise in immature hematopoietic progenitors, primarily peripheral blood mononuclear cells (PBMCs), following surgical trauma. Gzrelak *et al.*, note that IL-8 induces progenitor cell mobilization properties, but do not elaborate on whether the IL-8 reduced inflammation following surgical trauma. Laterveer *et al.*, teach the induction of hematopoietic progenitor cells after injection of hematopoietic growth factors, including G-CSF, GM-CSF, and SCF, followed by IL-8. Laterveer *et al.*, also teach that IL-8 creates a 17-fold increase in the number of circulating progenitor cells following saline injection in controls 15 minutes after injection (p. 1389, column 1, second paragraph and Figure 2, p. 1391). IL-8 is best known in the art as being chemoattractive for neutrophils, recruiting these phagocytic polymorphonuclear cells to sites of injury or tissue damage. It is well known that IL-8 is involved in granulocyte mobilization. IL-8 is considered to be inherently proinflammatory specifically because it recruits neutrophils to the sites of injury or trauma. Neither Gzrelak *et al.*, nor Laterveer *et al.*, contradict this well known fact. The recruitment of neutrophils to the site of myocardial infarct-related damage is a proinflammatory response because once activated, neutrophils and macrophages must move through the tissues to the site of trauma or injury in order clean up necrotic and apoptotic debris (see i.e. Kukielka *et al.*, J Clin Invest. 1995 Jan 95:89-103). Kukielka *et al.*, support the hypothesis that IL-8 participates in neutrophil-mediated myocardial injury by demonstrating the proinflammatory effects of IL-8 in the myocardium after ischemia and reperfusion.

11. The rejection is maintained because Applicant is not fully enabled for administration of IL-8 or other proinflammatory cytokines to reduce infarct-related myocardial tissue damage, including neurotrophic factors, listed in the Markush group of claim 5, for example. Brain-derived neurotrophic factor (BDNF), which belongs to the family of neurotrophins, has been shown to induce the expression of tachykinins, which are not beneficial to the myocardium following reperfusion injury. Hiltunen, et al. (J Pathol. 2001 Jun;194(2):247-53) teach BDNF in the pathogenesis of reperfusion injury and in the alterations of cardiac sensory and sympathetic neuronal function after myocardial ischemia and reperfusion (see p. 251: column 2: middle of first paragraph). The rejection is maintained for the reasons herein and for the reasons of record filed in the office actions of 03/22/2005 and 10/20/2004.

*35 USC § 102(b)*

12. The rejection of claims 1, 2, and 9 under 35 U.S.C. 102(b) as anticipated by Orlic *et al.* is maintained for reasons of record in the office action of 03/22/2005 and 10/20/2004. Applicant's arguments filed 12 August 2005, have been fully considered but they are not persuasive.

13. Applicants reiterate their position that both Orlic *et al.*, and Anversa *et al.*, did not teach the instant invention as claimed. Applicants argue that both Orlic *et al.*, and Anversa *et al.*, administered their compositions comprising G-CSF prophylactically, prior to the occurrence of a myocardial infarction, and then again after the occurrence of a myocardial infarction. Thus, Applicants argue, that Orlic *et al.*, and Anversa *et al.*, teach an additional step, which is not required in the instant method. Applicants also argue that they have distinguished the presently-claimed subject matter in the context of steps of treatment, through the use of the term "consisting of" and that no need exists to further limit the claimed subject matter to use of compositions having no other component than G-CSF.

14. The claims recite an improvement on a method to reduce infarct-related myocardial tissue damage. Applicants argue that Orlic *et al.* teach mobilization of bone marrow cells "prior to acute myocardial infarction (AMI)". Orlic *et al.* also teach the administration of a composition comprising G-CSF three days following coronary artery ligation, demonstrating that this composition was given following ischemia and not merely prophylactically (p. 10344:column 2:line 16 and p. 10349:column 2:lines 5-7).

15. Applicants misunderstand the Examiner's concern over the use of open language in the term "comprising." The amendment of the claim 1 to read "consisting of administering" does not resolve the application of prior art to this claim because Applicants maintain the use of open language in the phrase "a composition comprising G-CSF." By amending the claim language of the improvement, the steps of the improvement are now limited to the specific steps disclosed. However, the prior art remains applicable. The claims read on a composition "comprising" G-CSF. The claims are drafted using open language that precludes any argument relating to the use of G-CSF alone.

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16. The rejection of claims 1-7 and 9-10 under 35 U.S.C. 102(b) as anticipated by Anversa *et al.*, is maintained for the reasons of record in the office action of 03/22/2005 and 10/20/2004.

17. The claims recite an improvement on a method to reduce infarct-related myocardial tissue damage. Anversa *et al.*, teach the administration of cytokines, including G-CSF for the treatment or therapy of infarct-related myocardial tissue damage at p. 1 (0006). The methods disclosed by Anversa *et al.* are drawn to treating cardiovascular diseases, including ischemia (the most common cause of which is myocardial infarction), and taking advantage of the regenerative properties of stem cells and cytokines that can restore cardiac function (p. 1: paragraph (0003) and p. 2: paragraph (0022), respectively). The methods disclosed by Anversa *et al.*, are also drawn to treating cardiovascular diseases, including ischemia, and define ischemic events as encompassing clinical scenarios such as bypass surgery (p. 1: paragraph (0003) and p. 2: (0014), respectively). Further, Anversa *et al.*, state that the administration of cytokines, including G-CSF, following ischemia involves neoangiogenesis and restores "structural and functional integrity to the infarcted area" (p. 3: paragraph (0038), p. 4: paragraph (0044)).

18. The methods taught by Orlic *et al.*, and Anversa *et al.*, have the same steps and the same starting materials (administration of a composition comprising G-CSF in an effective amount in a mammal suffering from ischemic vascular diseases including ischemic cardiomyopathy or myocardial ischemia) as the presently claimed methods. Any additional steps taught by Orlic *et al.*, or Anversa *et al.*, are not limiting as applied to the instant claims. So long as Orlic *et al.*, and Anversa *et al.*, teach each of the steps of the instant claimed methods (which they do), their teachings fully encompass the claimed methods herein. As such, the presently claimed methods are not distinguishable from the methods taught by Orlic *et al.*, and Anversa *et al.*

***New Claim Rejections – Necessitated by Amendment***

***Claim Rejections - 35 USC § 112, First Paragraph***

20. Claims 11-16 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. This is a new matter rejection. The claims contain subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention. This is a new matter rejection. Claims 11-16 recite the method of claim 1 wherein the reduction in damage is characterized by an improvement in cardiac function, reduced scarring of the myocardium, reduced



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cardiomyocyte apoptosis, reduced necrosis, regeneration of the myocardium, and induced neoangiogenesis in the infarcted zone. The specification discloses the teachings of Kocher *et al.* (Nature Med. 7:430-436, 2001) (see specification at p. 6, lines 14-19), wherein Kocher *et al.*, demonstrated that the intravenous injection of adult human bone-marrow-derived endothelial cell precursors, mobilized by treatment with G-CSF, into a rat model of myocardial ischemia induced neoangiogenesis in the infarcted zone; prevented cardiomyocyte apoptosis, reduced scar formation, and improved ventricular function. Kocher *et al.*, realized the reduction in damage by using bone-marrow stem cells that had been pre-treated with G-CSF. The treated, activated cells were then injected into the subjects. However, the methodologies of Kocher *et al.*, are different than the method claimed in the instant application. Applicant's are claiming improvements in a method of direct administration of G-CSF before, concurrently with, and/or after reperfusion therapy. Thus, claims 11-16 represent new matter that is not described in the specification.

**35 USC § 112 – Second Paragraph**

21. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

22. Claims 1-7, 9, and 11-16 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. The claims are drawn to the improvement of a method of reperfusion therapy for treating acute myocardial infarction in a mammal to reduce infarct-related myocardial tissue damage, the improvement consisting of administering an effective amount of a composition comprising Granulocyte Colony Stimulating Factor (G-CSF) polypeptide after AMI, but before, concurrently with, and or after reperfusion therapy and an improvement in a method of reperfusion therapy for treating occlusion in an artery in a mammal to reduce tissue damage. The claims teach methods of improvement of reperfusion therapy. However, the instant claims are drawn to administration before, with, and/or after reperfusion therapy, which extends the method outside the reperfusion therapy. It is unclear from the language of the claims whether the applicant is claiming an improvement on a method of reperfusion therapy (thereby necessarily limiting the improvement to the metes and bounds of the reperfusion therapy) or whether the applicant is claiming a method of treatment before, during, and/or after reperfusion therapy.

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23. Claim 10 is rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. The claims are drawn to an improvement in a method of bypass surgery for treating occlusion in an artery in a mammal to reduce tissue damage, the improvement consisting of administering an effective amount of G-CSF. However, the instant claims are drawn to administration before, with, and/or after bypass surgery, which extends the method outside the bypass surgery. It is unclear from the language of the claims whether the applicant is claiming an improvement on a method of bypass surgery (thereby necessarily limiting the improvement to the metes and bounds of the bypass surgery) or whether the applicant is claiming a method of treatment before, during, and/or after bypass surgery.

24. A broad range or limitation together with a narrow range or limitation that falls within the broad range or limitation (in the same claim) is considered indefinite, since the resulting claim does not clearly set forth the metes and bounds of the patent protection desired. See MPEP § 2173.05(c). Note the explanation given by the Board of Patent Appeals and Interferences in *Ex parte Wu*, 10 USPQ2d 2031, 2033 (Bd. Pat. App. & Inter. 1989), as to where broad language is followed by "such as" and then narrow language. The Board stated that this can render a claim indefinite by raising a question or doubt as to whether the feature introduced by such language is (a) merely exemplary of the remainder of the claim, and therefore not required, or (b) a required feature of the claims. Note also, for example, the decisions of *Ex parte Steigewald*, 131 USPQ 74 (Bd. App. 1961); *Ex parte Hall*, 83 USPQ 38 (Bd. App. 1948); and *Ex parte Hasche*, 86 USPQ 481 (Bd. App. 1949). In the present instance, claim 5 recites the broad recitation interleukins, and the claim also recites IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, and IL -12 which is the narrower statement of the range/limitation.

***New Claim Rejections – Necessitated by Amendment***  
***35 USC § 102***

25. Claims 11-16 are rejected under 35 U.S.C. 102(b) as being anticipated by Orlic *et al.* (PNAS 2001, previously cited in Office Action of 03/22/2005 and 10/20/2004), as evidenced by Gottlieb *et al.* (Ann N Y Acad Sci. 1999 Jun 30;874:412-26). The claims recite improvements in a method of reperfusion therapy where the reduction in damage is characterized by improvement in cardiac function, reduced scarring of the myocardium, reduction in cardiomyocyte apoptosis, reduction in necrosis, regeneration of the myocardium, and neoangiogenesis in the infarcted zone. Orlic *et al.*, teach improvement in cardiac function of mice after infarct-related myocardial tissue damage and treatment

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with G-CSF (p. 10347:column 2:first full paragraph), myocardial regeneration after infarct-related myocardial tissue damage and treatment with G-CSF (p. 10345:column 2:first full paragraph; p. 10347:column 1:second paragraph; and Figure 2, p. 10346), reduced scarring (encompassing reduced necrosis and apoptosis) of the myocardium after infarct-related myocardial tissue damage and treatment with G-CSF (p. 10345:column 2:first full paragraph; p. 10348:column 1:first paragraph; and Figure 1C), neoangiogenesis in the infarcted zone after infarct-related myocardial tissue damage and treatment with G-CSF (p. 10346:column 2:first paragraph). Cardiomyocyte necrosis and apoptosis are inherent properties of scarring resulting from cardiac infarction (see i.e. Gottlieb *et al* Review, specifically, p. 412, abstract, line 10, and p.423, first full paragraph).

26. Claims 11-16 are rejected under 35 U.S.C. 102(b) as anticipated by Anversa *et al.* (US 2002/0061587 A1, published May 23, 2002, previously cited in the Office Action of 3/22/2005 and 10/20/2004). The instant claims recite improvements in a method of reperfusion therapy where the reduction in damage is characterized by improvement in cardiac function, reduced scarring of the myocardium, reduction in cardiomyocyte apoptosis, reduction in necrosis, regeneration of the myocardium, and neoangiogenesis in the infarcted zone. Anversa *et al.*, teach the administration of cytokines, including G-CSF for the treatment or therapy of infarct-related myocardial tissue damage in several species of mammals at p. 1: paragraph (0006). The methods disclosed by Anversa *et al.* are drawn to treating cardiovascular diseases, including ischemia (the most common cause of which is myocardial infarction), and taking advantage of the regenerative properties of stem cells and cytokines that may restore cardiac function (p. 1: paragraph (0003) and p. 2: paragraph (0022), respectively). Furthermore, Anversa *et al.*, state that the administration of cytokines, including G-CSF, following ischemia involves neoangiogenesis and restores "structural and functional integrity to the infarcted area" (p. 3: paragraph (0038)). Myocardial regeneration and reduced scar formation (encompassing reduced necrosis and apoptosis) in the cytokine-treated mice is discussed (p 15: paragraph (0180)).

**NO CLAIMS ARE ALLOWED.**

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Cherie M. Chappell whose telephone number is (571) 272-3329. The examiner can normally be reached on Monday - Thursday 9:00am-7:30pm (EST).

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If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Brenda Brumback can be reached on (571) 272-0961. The fax phone number for the organization where this application or proceeding is assigned is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

CMC

  
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